# Hepatitis B Virus

Note: Serological markers for different forms of HBV infection

1. Acute infection: HBsAg, IgM HBcAb, HBeAg
2. Chronic persistent carrier (asymptomatic): HbsAg, IgG HBeAb
3. Chronic active hepatitis (symptomatic, infectious): HBsAg, HBeAg
4. Past exposure to HBV: IgG HBsAb, HBcAb
5. successful immunization: only see much IgG HBsAb

Presentation:

1. 27 yr old woman- fevers, chills, headache, malaise, anorexia, abdominal pain for days.
2. came in because her eyes turned yellow, developed generalized itching
3. admitted to IV drug use for past few years, shared needles with friends
4. mild distress due to pruritis (bothersome itch), scleral icterus, jaundice present. Liver enlarged / mildly tender
5. elevated lymphocytes. ALT 2730, AST 2390
6. Serology: Hepatitis panel- detected HBV surface Antigen = HBsAg in serum + found IgM for HBcAb

Hepatitis B virus (HBV)

1. double shelled DNA virus in Hepadnaviridae family
2. genome is circular double stranded DNA
3. 3 different particles seen:
   1. larger rounded bodies = full assembly infectious virus with lipid envelope
   2. 2 smaller particles = spherical nucleocapid core and tubular / filamentous particles of surface proteins (HbsAg). These are produced as decoys to evade the immune system.
   3. HBsAg: Hepatitis B surface Ag – the envelope protein expressed on outer surface of full virion and on smaller particles
4. HBsAg: primary component for HBV vaccine- long-term protection
5. inner core of virus contains HB core antigen (HBcAg), Be Ag DNA, and DNA-dependent DNA polymerase (with reverse transcriptase function)
6. HBsAG, HBcAg, HBeAg and specific Ab are the diagnostic markers
7. does not grow in cell cultures, but HBV DNA can transfect surrogate cells to produce viral proteins

Epidemiology

1. Transmission in humans: percutaneous or parenteral inoculation (IV drug use) or permucosal exposure to fluids (sexual contact) or by Perinatal exposure (maternal-neonatal; vertical)
2. Most in US is by sexual contact
3. any sort of piercing or needle sticks can be a source- but are only small portion of cases in US

Pathogenesis:

1. incubation ranges from 45 days to 210 days for Jaundice
2. After entry🡪 virus becomes blood-borne🡪 produces sustained viremia
3. Replicates in hepatocytes with minimal cytopathic effect, can replicate for a long time without liver damage
4. virus specific cytotoxic T lymphocytes are responsible for clinical manifestations and for eventual resolution of infection
5. onset of acute disease is insidious
6. most acute HBV infections result in complete recovery
7. Markers:
   1. First marker to appear: HBsAg – detect 1-2 weeks after exposure, no longer seen after recovery after 3 months
   2. Anti-HBs seen after disappearance of HBsAg
   3. HBeAg is transiently detected with acute infection
   4. IgM to Hepatitis B core Ag (IgM HBcAb) in serum is detectable at time of clinical onset🡪 declines to undetectable after 6 months
   5. IgG HBcAb persists as marker of past infection
   6. Diagnosis of acute HBV infection can be made based on detection of IgM HBcAb

Treatment: no treatment, management is supportive. May use antivirals for chronic infection

Prevention:

1. avoid risk factors suck as multiple sexual partners and IV drug use
2. beware of needle sticks
3. don’t be a whore or a druggie
4. can use Ig for post exposure prophylaxis
5. Recombinant vaccine is very effective and safe- also useful for preventing infection in recently exposed individual.